a reentrant circuit was present in the RA free wall; and 4) exhibited a line of functional block in the RA free wall.

982-122 Multichannel Mapping of Atrial Activation Sequences After Internal Atrial Defibrillation Shocks

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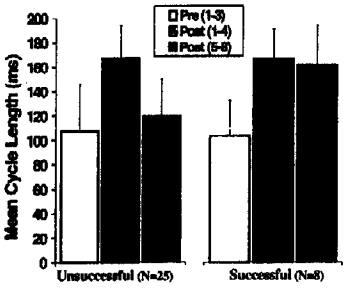
The purpose of this study was to characterize the patterns of atrial activation before and after internal atrial defibrillation shocks in a model of atrial fibrillation (AF). In 5 adult sheep, unipolar electrograms were recorded from 336 epicardial and 24 endocardial electrodes on the right atrial septum before and after shocks. After AF induction with rapid atrial pacing, biphasic shocks (3/3 ms) near threshold were delivered between transvenous electrodes in the right atrial appendage and distal coronary sinus. Activation sequences (AS) and mean cycle lengths (CL) for the 3 cycles before the shock [Pre (1–3)], the first 4 [(Post (1–4)], and the second 4 [Post (5–8)] cycles after the shock were determined for successful and unsuccessful shocks (Fig.).



For unsuccessful shocks (N = 25), slower, more organized AS for Post (1–4) propagated over the entire atria; AS became less organized and faster for Post (5–8). The mean CL of the Post (1–4) were longer than Pre (1–3) and Post (5–8) ($p \leq 0.05$). For successful shocks (N = 25) type A and B AS were seen. Type A (N = 17) was characterized by lack of atrial fibrillatory activity after the shock and type B (N = 8) showed continued post-shock activations of both atria, with AS becoming slower and more organized with eventual termination after 6 ± 2 cycles. The mean CL of Post (1–4) were longer than Pre (1–3) ($p \leq 0.05$). but were not significantly longer than Post (5–8). Successful and unsuccessful internal atrial defibrillation shocks alter AS and post-shock AS may predict whether a shock will fail or succeed (Type B).

982-123 Mechanism of Onset of Induced Sustained Atrial Fibrillation in the Canine Sterile Pericarditis Model

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Prior mapping studies during induced, sustained atrial fibrillation (AF) in the canine sterile pericarditis model showed that multiple unstable reentrant circuits which involve the septum and the atrial epicardium are responsible for maintaining AF. These reentrant circuits cyclically disappear while others reform principally from wave fronts coming from the septum. To test the hypothesis that the initial formation of an unstable septal reentrant circuit generates the wave fronts which develop multiple unstable reentrant circuits, we performed simultaneous multisite mapping from both atria including the atrial septum during the onset of induced AF. Activation patterns were recorded with 388 electrodes during 7 episodes of AF, 5 sustained (≥5 min) and 2 nonsustained AF (<5 min), in 7 dogs. AF was induced by programmed atrial stimulation or rapid atrial pacing from the selected atrial sites. Activation maps were analyzed during the atrial pacing and for a 1 sec period after cessation of pacing during the onset of AF. In all episodes, wave fronts produced by atrial pacing always developed an unstable reentrant circuit which involved the septum and atrial epicardium. This septal reentrant circuit generated wave fronts, which developed other unstable reentrant circuits, so that during the 1 sec period of analysis, 13–19 (mean 16 ± 7) unstable reentrant circuits were formed. Their mean cycle length was 96 ± 5 ms and their mean number of consecutive rotations was 2.9 ± 0.8. These reentrant circuits were located principally involving the septum and the right atrial free wall, but also sometimes in the left atrium. In contrast, 2 episodes of nonsustained AF had only 8–9 (mean 8.5) unstable reentrant circuits with longer cycle lengths (mean 125 ms) which primarily involved the septum and atrial epicardium, but rarely were entirely located in the right or left atrium. *Conclusion:* Induced, sustained AF in this model occurs after the initial formation of an unstable septal reentrant circuit, followed by the development of multiple unstable reentrant circuits with very short cycle length.

982-124 Transmembrane Potential Properties of Cells at the Core of Reentry in Isolated Canine Right Atria

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The characteristics of transmembrane action potential (TMP) properties at the core of reentrant activity in cardiac tissue remain poorly defined. In 5 isolated perfused canine right atria, we performed endocardial mapping using a 480 electrode plaque (1.6 mm inter-bipole distance). Simultaneous TMP recordings were made from the epicardial surface. 61 episodes of reentry, identified by electrogram analysis and computerized dynamic activation display, were induced by S1–S2 protocol in the presence of 1 to 5 μM of acetylcholine. Successful simultaneous TMP recordings and activation maps were made in 6 of 61 episodes of induced reentry. The core was identified by the pivot point of reentry and marked by the absence of activation or double potentials on the endocardial map. We defined TMP recordings to be "near the core" if it is within 3.2 mm from the core, otherwise, they are "in the periphery". A total of 115 beats were analyzed including 60 near the core and 55 in the periphery. The mean cycle length of reentry was 127.0 ± 17.1 ms. The action potential amplitude, duration (90% repolarization) and dV/dt max near the core were 55.7 ± 26.3 mV, 56.2 ± 21 ms and 28.2 ± 21.8 V/s, respectively, significantly less than those in the periphery 75.4 ± 3.5 mV, 64.7 ± 5.4 ms and 45.6 ± 9.8 V/s ($p < 0.001$, < 0.01, < 0.001, respectively). In 2 episodes of reentry, the cell at the core remained unexcited at its resting membrane potential (RMP). We conclude that during reentry: 1) Action potentials of cells near the core have a reduced amplitude, duration and dV/dt max. 2) Cells at the core remain quiescent at their RMP. These findings are compatible with reentry caused by spiral waves of excitation.

982-125 An Unexpected Pattern of Anterior AV Nodal Input ('Fast Pathway') and Atrial Septal Activation During Sinus Rhythm

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Background: It is assumed that activation of the low interatrial septum (IAS) occurs anterogradely from the sinus node (SN). *Objective:* To determine the activation pattern of a SN impulse in the rabbit right atrium with special attention to the sequence of excitation of the anterior ('fast pathway', FP) and posterior ('slow pathway', SP) atrioventricular (AV) nodal input regions. *Methods:* Superfused right atrial preparations, containing the SN and AV node, crista terminalis (CrT) and IAS from 5 New Zealand White rabbits were mapped during SN rhythm using bipolar electrodes. A thermoelectric cooling probe with a 3.75 mm^2 tip (Novoste Corp.) was used to cool discrete areas of the right atrium up to 16ºC. Brief AV nodal postganglionic vagal stimulation was used to induce transient nodal block. *Results:* During SN rhythm the high CrT and SP input regions were activated rapidly (SN-high CrT time = 2.2 ± 1 ms and SN-SP time = 18 ± 8 ms) compared to the high IAS and FP input regions, respectively (SN-high IAS time = 41 ± 14, $p < 0.05$ and SN-FP time = 34 ± 11 ms, $p < 0.05$) in all preparations. Electrograms containing 2 distinct components were recorded from the IAS in 4 preparations. Cooling up to 16ºC along the tendon of Todaro increased the time interval between excitation of SP and FP in 4 preparations. Induction of AV nodal block did not alter the above activation sequence. In addition, the cooling increased the interval between the 2 components of the IAS electrograms in the same preparations. *Conclusions:* 1. The CrT is engaged more rapidly than the IAS from the SN. 2. Activation of the fast pathway input region can depend on, and follow activation of the slow pathway input region of the AV node via a route along the tendon of Todaro. 3. Such an activation pattern is unexpected, requires evaluation in normal humans and those with sick sinus syndrome, and needs to be considered in models of dual AV nodal physiology.

982-126 Dual AV Nodal Physiology Without Inducible Tachycardia in Patients With Spontaneous, Documented Tachycardia – Clinical Characteristics, and the Results of Selective Radiofrequency Catheter Ablation of the Slow Pathway

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Sustained AV nodal reentrant tachycardia (AVNRT) sometimes cannot be induced despite the presence of dual AV nodal physiology and documentation of clinical tachycardia. The clinical characteristics and the role of radiofrequency catheter ablation (RFCA) in this group of pts have not been clearly defined. We examined a total of 19 consecutive pts with similar presentations. There were 13 men (68%) and 6 women, and the mean age was 35 ±